Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSSPTA1654MCG

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	DEC 23	New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/ USPAT2
NEWS 4	JAN 13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS 5	JAN 13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
NEWS 6	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS 7	JAN 17	IPC 8 in the WPI family of databases including WPIFV
NEWS 8	JAN 30	Saved answer limit increased
NEWS 9	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS 10	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS 11	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS 12	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 13	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS 14	FEB 28	TOXCENTER reloaded with enhancements
NEWS 15	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS 16	MAR 01	INSPEC reloaded and enhanced
NEWS 17	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS 18	MAR 08	X.25 communication option no longer available after June 2006
NEWS 19	MAR 22	EMBASE is now updated on a daily basis
NEWS 20	APR 03	New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS 21	APR 03	Bibliographic data updates resume; new IPC 8 fields and IPC thesaurus added in PCTFULL
NEWS 22	APR 04	STN AnaVist $500 visualization usage credit offered
NEWS 23	APR 12	LINSPEC, learning database for INSPEC, reloaded and enhanced
NEWS 24	APR 12	Improved structure highlighting in FQHIT and QHIT display in MARPAT
NEWS 25	APR 12	Derwent World Patents Index to be reloaded and enhanced during second quarter; strategies may be affected

NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
http://download.cas.org/express/v8.0-Discover/

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation

of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 11:12:40 ON 17 APR 2006

=> file medline embase
COST IN U.S. DOLLARS SINCE FILE ENTRY 0.21 TOTAL SESSION 0.21
FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 11:12:51 ON 17 APR 2006

FILE 'EMBASE' ENTERED AT 11:12:51 ON 17 APR 2006

=> "cartilage atrophy"
L1 33 "CARTILAGE ATROPHY"

=> l1 and pain
L2 0 L1 AND PAIN

=> l1 and osteoarthrosis
L3 0 L1 AND OSTEOARTHROSIS

=> l1 and osteoarthritis
L4 10 L1 AND OSTEOARTHRITIS

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 5 DUP REM L4 (5 DUPLICATES REMOVED)

=> d ibib abs total

L5 ANSWER 1 OF 5 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2003591337 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14673989
TITLE: Longitudinal analysis of **cartilage atrophy** in the knees of patients with spinal cord injury.
AUTHOR: Vanwanseele B; Eckstein F; Knecht H; Spaepen A; Stussi E
CORPORATE SOURCE: Swiss Federal Institute of Technology, Zurich, Switzerland.. vanwanseele@biomech.mat.ethz.ch
SOURCE: Arthritis and rheumatism, (2003 Dec) Vol. 48, No. 12, pp. 3377-81.
Journal code: 0370605. ISSN: 0004-3591.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200401
ENTRY DATE: Entered STN: 20031216
Last Updated on STN: 20040115
Entered Medline: 20040114
AB OBJECTIVE: A previous cross-sectional study indicated that the morphology of patellar and tibial cartilage is subject to change after spinal cord injury (SCI). The aim of this study was to perform a longitudinal analysis of **cartilage atrophy** in all knee compartments, including the femoral condyles, in SCI patients over 12 months. METHODS: The right knees of 9 patients with complete, traumatic SCI were examined shortly after the injury (mean +/- SD 9 +/- 4 weeks) and at 6 and 12 months postinjury. Three-dimensional morphology of the patellar, tibial, and femoral cartilage (mean and maximum thickness, volume, and surface area) was determined from coronal and transversal

magnetic resonance images (fat-suppressed gradient-echo sequences) using validated postprocessing techniques. RESULTS: The mean thickness of knee joint cartilage decreased significantly during the first 6 months after injury (range 5-7%; P < 0.05). The mean change at 12 months was 9% in the patella, 11% in the medial tibia, 11% in the medial femoral condyle, 13% in the lateral tibia, and 10% in the lateral femoral condyle (P < 0.05 for all compartments). CONCLUSION: This is the first report of a longitudinal analysis of **cartilage atrophy** in patients with SCI.
These data show that human **cartilage atrophies** in the absence of normal joint loading and movement after SCI, with a rate of change that is higher than that observed in **osteoarthritis** (OA).
A potential clinical implication is that cartilage thinning after SCI may affect the stress distribution in the joint and render it vulnerable to OA. Future studies should focus on whether specific exercise protocols and rehabilitation programs can prevent cartilage thinning.

L5 ANSWER 2 OF 5 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 92208668 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1555051
TITLE: A mini review: proteoglycan aggregate profiles in the Pond-Nuki dog model of **osteoarthritis** and in canine disuse atrophy.
AUTHOR: Howell D S; Muller F; Manicourt D H
CORPORATE SOURCE: Department of Medicine, University of Miami School of Medicine, FL 33101.
SOURCE: British journal of rheumatology, (1992) Vol. 31 Suppl 1, pp. 7-11. Ref: 24
Journal code: 8302415. ISSN: 0263-7103.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199205
ENTRY DATE: Entered STN: 19920515
Last Updated on STN: 19920515
Entered Medline: 19920507
AB The Pond-Nuki dog model of **osteoarthritis** has characteristics which seem to mimic the human disease in early stages, particularly with respect to progressive changes in the cartilage matrix. Aggregating proteoglycans were studied using novel extraction and ultracentrifugation methods designed to separate very large macromolecules. With these methods two large peaks of proteoglycan (PG) aggregates (PGA-1 and PGA-2) were separated in preparative amounts and were shown to have unequivocal differences in composition in many respects. The profiles of these peaks have been studied as a function of joint location, topographic site, cartilage layer, presence of **cartilage atrophy** versus **osteoarthritis**, as well as treatment of the animals with various agents. Both link protein (essential for forming link-protein stabilized aggregates) and hyaluronate are required to regenerate normal aggregate profiles from the deficient aggregate fractions obtained from osteoarthritic cartilage. Canine proteoglycan link-stabilized aggregates (PGA-2) are confined to the middle and deep zone of cartilage. We believe that their reduction or elimination in the Pond-Nuki model results from a disturbance or loss of functional link protein (and hyaluronate), thereby weakening the middle and deep cartilage layers.

L5 ANSWER 3 OF 5 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 90037565 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2808699
TITLE: Cartilage matrix glycoprotein is present in serum in experimental canine **osteoarthritis**.
AUTHOR: Fife R S; Brandt K D
CORPORATE SOURCE: Department of Medicine, Indiana University School of Medicine, Indianapolis 46202.

CONTRACT NUMBER: AR-20582 (NIAMS)
AR-34367 (NIAMS)
AR-39250 (NIAMS)
SOURCE: The Journal of clinical investigation, (1989 Nov) Vol. 84, No. 5, pp. 1432-9.
Journal code: 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198912
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19970203
Entered Medline: 19891204

AB We have described previously a disulfide-bonded 550,000-D cartilage matrix glycoprotein (CMGP), which is found in normal hyaline cartilage, fibrocartilage, and the vitreous of the eye, and consists of subunits with apparent molecular weights of 130,000 in 4% gels (116,000 in 9% gels). In osteoarthritic cartilage from dogs subjected to transection of the anterior cruciate ligament (ACL), CMGP is cleaved to major immunoreactive fragments with apparent molecular weights of 65,000 and 75,000 after reduction with 2-mercaptoethanol. In the present study, using immunolocation analysis, a monoclonal antibody to CMGP did not react with serum from 8 of 12 dogs before ACL transection but did react with serum from seven of these animals 4 wk after surgery and with serum from 10 dogs at sacrifice, 8-14 wk after ACL transection. Serum from four dogs reacted with the monoclonal antibody before ACL transection. Serum from two dogs was negative at all time points. Immunolocation studies using a polyclonal antiserum to CMGP were performed in seven of these dogs and produced results identical with the monoclonal antibody in four dogs. In contrast, analysis of serial serum samples from three dogs with **cartilage atrophy** revealed no evidence of CMGP at any time point. These data suggest that CMGP may be a serum marker for **osteoarthritis** in this canine model.

L5 ANSWER 4 OF 5 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 85045915 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6497604
TITLE: Synovectomy as treatment for purulent joint infection.
AUTHOR: Tscherne H; Giebel G; Muhr G; Howell C
SOURCE: Archives of orthopaedic and traumatic surgery. Archiv fur orthopadische und Unfall-Chirurgie, (1984) Vol. 103, No. 3, pp. 162-4.
Journal code: 7803037. ISSN: 0344-8444.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198412
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19841220

AB Conventional treatment of pyogenic knee joint infections leads to unsatisfactory results. Through early synovectomy, before cartilage damage and **osteoarthritis** appear, the infected focus can be "excised." Functional after-treatment avoids **cartilage atrophy**, wound adhesions, and muscle weakness. The excellent results after 26 knee joint infections confirm this.

L5 ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 84277727 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6465163
TITLE: Effects of salicylates and other nonsteroidal anti-inflammatory drugs on articular cartilage.
AUTHOR: Brandt K D; Palmoski M J

CONTRACT NUMBER: AM 20582 (NIADDK)
AM 27075 (NIADDK)
SOURCE: The American journal of medicine, (1984 Jul 13) Vol. 77, No. 1A, pp. 65-9.
Journal code: 0267200. ISSN: 0002-9343.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198409
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19840907

AB According to in vivo experimental data, salicylates and several other nonsteroidal anti-inflammatory agents suppress proteoglycan biosynthesis in normal and degenerating articular cartilage. Therapeutic levels of aspirin in vivo had a similar adverse effect on degenerating cartilage, as noted in two canine models of **osteoarthritis** and **cartilage atrophy**. Because the effective daily antirheumatic dose of nonsteroidal anti-inflammatory drugs is lower than that of salicylates, these drugs may have less negative effects on degenerating articular cartilage. However, clinical significance cannot be extrapolated from these experimental data.

=> d ibib abs total l1

L1 ANSWER 1 OF 33 MEDLINE on STN
ACCESSION NUMBER: 2004427822 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15334465
TITLE: **Cartilage atrophy** in the knees of patients after seven weeks of partial load bearing.
AUTHOR: Hinterwimmer S; Krammer M; Krotz M; Glaser C; Baumgart R; Reiser M; Eckstein F
CORPORATE SOURCE: Ludwig-Maximilians-Universitat Munchen, Munich, Germany.
SOURCE: Arthritis and rheumatism, (2004 Aug) Vol. 50, No. 8, pp. 2516-20.
Journal code: 0370605. ISSN: 0004-3591.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200409
ENTRY DATE: Entered STN: 20040831
Last Updated on STN: 20040925
Entered Medline: 20040924

AB OBJECTIVE: It is currently unknown whether human cartilage properties change during short periods of partial load bearing. We used a post-ankle fracture model to explore whether changes in cartilage morphology occur in the knee under conditions of partial load bearing. METHODS: The knees of 20 patients with Weber type B and type C fractures were examined using magnetic resonance imaging. The first scan was obtained shortly (mean +/- SD 3.2 +/- 3.0 days) after the injury, and a second scan was obtained 7 weeks later (mean +/- SD 50.7 +/- 5.5 days). The morphology (mean and maximum thickness, volume, and surface area) of the patellar, tibial, and femoral cartilage was determined from coronal and axial magnetic resonance images (fat-suppressed gradient-echo). RESULTS: Between week 0 and week 7, the cross-sectional area of the quadriceps muscle was reduced by 11% (P< 0.001). Changes in the mean (+/-SD) cartilage thickness ranged from -2.9 +/- 3.2% in the patella to -6.6 +/- 4.9% in the medial tibia. No significant change in cartilage morphology of the contralateral knee was observed. CONCLUSION: Results of this study demonstrate that in a post-ankle fracture model of partial load bearing, cartilage morphology in all knee compartments is subject to significant change. Changes in the femorotibial joint exceeded those in the patella, whereas no change was

observed in the contralateral knee. These findings raise the question of whether cartilage is mechanically less competent and particularly vulnerable after states of partial or complete immobilization.

L1 ANSWER 2 OF 33 MEDLINE on STN
ACCESSION NUMBER: 2003591337 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14673989
TITLE: Longitudinal analysis of **cartilage atrophy** in the knees of patients with spinal cord injury.
AUTHOR: Vanwanseele B; Eckstein F; Knecht H; Spaepen A; Stussi E
CORPORATE SOURCE: Swiss Federal Institute of Technology, Zurich, Switzerland.. vanwanseele@biomech.mat.ethz.ch
SOURCE: Arthritis and rheumatism, (2003 Dec) Vol. 48, No. 12, pp. 3377-81.
Journal code: 0370605. ISSN: 0004-3591.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200401
ENTRY DATE: Entered STN: 20031216
Last Updated on STN: 20040115
Entered Medline: 20040114

AB OBJECTIVE: A previous cross-sectional study indicated that the morphology of patellar and tibial cartilage is subject to change after spinal cord injury (SCI). The aim of this study was to perform a longitudinal analysis of **cartilage atrophy** in all knee compartments, including the femoral condyles, in SCI patients over 12 months. METHODS: The right knees of 9 patients with complete, traumatic SCI were examined shortly after the injury (mean +/- SD 9 +/- 4 weeks) and at 6 and 12 months postinjury. Three-dimensional morphology of the patellar, tibial, and femoral cartilage (mean and maximum thickness, volume, and surface area) was determined from coronal and transversal magnetic resonance images (fat-suppressed gradient-echo sequences) using validated postprocessing techniques. RESULTS: The mean thickness of knee joint cartilage decreased significantly during the first 6 months after injury (range 5-7%; $P < 0.05$). The mean change at 12 months was 9% in the patella, 11% in the medial tibia, 11% in the medial femoral condyle, 13% in the lateral tibia, and 10% in the lateral femoral condyle ($P < 0.05$ for all compartments). CONCLUSION: This is the first report of a longitudinal analysis of **cartilage atrophy** in patients with SCI. These data show that human **cartilage atrophies** in the absence of normal joint loading and movement after SCI, with a rate of change that is higher than that observed in osteoarthritis (OA). A potential clinical implication is that cartilage thinning after SCI may affect the stress distribution in the joint and render it vulnerable to OA. Future studies should focus on whether specific exercise protocols and rehabilitation programs can prevent cartilage thinning.

L1 ANSWER 3 OF 33 MEDLINE on STN
ACCESSION NUMBER: 2003050918 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12560716
TITLE: Reinforced orbitotemporal lift: contribution to midface rejuvenation.
AUTHOR: Reno Waldir Teixeira
CORPORATE SOURCE: Plastic Surgery Service at Santa Casa, Misericordia de Guaratingueta Hospital, Sao Paulo, Brazil.
SOURCE: Plastic and reconstructive surgery, (2003 Feb) Vol. 111, No. 2, pp. 869-77; discussion 878-9.
Journal code: 1306050. ISSN: 0032-1052.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20030202
Last Updated on STN: 20030227
Entered Medline: 20030226

AB The changes in the aging face occur from progressive ptosis of the skin, fat, and muscle, in conjunction with bone absorption and **cartilage atrophy**. In the orbital region, hollowness and compartmentalization occur. Conventional face lift procedures correct only the skin flaccidity, and superficial musculoaponeurotic system techniques reposition the skin and platysma without repositioning the middle third of the face, creating an artificial jawline. Subperiosteal rhytidectomy disrupts the anatomy of the periorbita, which gives the patient a certain scarecrow aspect. Composite rhytidectomy associated with brow lift and blepharoplasty may offer better results, with improvement in the malar and orbital regions. The reinforced orbitotemporal lift (ROTEL) is a new procedure in a face lift that allows the orbicularis oculi muscle and all the structures connected to it to be elevated and stretched and the orbitotemporal skin to be raised, repositioning these structures and ending orbital compartmentalization. The result is an impressive improvement in the malar-orbitotemporal region, resulting in a natural and youthful appearance.

L1 ANSWER 4 OF 33 MEDLINE on STN
ACCESSION NUMBER: 2000204118 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10738181
TITLE: Bronchial **cartilage atrophy** in chronic bronchitis: observations on chondrolytic processes.
AUTHOR: Tetlow L C; Freemont A J; Woolley D E
CORPORATE SOURCE: University Department of Medicine, Manchester Royal Infirmary, Manchester, UK.. lynne.c.tetlow@man.ac.uk
SOURCE: Pathobiology : journal of immunopathology, molecular and cellular biology, (1999 Jul-Aug) Vol. 67, No. 4, pp. 196-201.
Journal code: 9007504. ISSN: 1015-2008.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000505
Last Updated on STN: 20000505
Entered Medline: 20000426

AB The status of bronchial cartilage degeneration in chronic bronchitis is unclear, and little is known about the chondrolytic mechanisms involved. The potential contributions of various inflammatory cells, chondrocytes and cartilage-degrading enzymes to **cartilage atrophy** have been examined. Bronchial cartilage specimens were obtained at autopsy from lobar secondary bronchi from chronic bronchitics and age-matched controls; each was examined by light microscopy and immunohistology for the distributions of mast cells, macrophages, eosinophils, collagenase 1, collagenase 3, and degradation products of cartilage collagen. Most bronchitic specimens showed hypertrophic chondrocytes, some of which were immunostained for collagenase 3, and occasionally for collagenase 1. Evidence for collagen degradation products was demonstrated around the lacunae of a proportion of chondrocytes, and both collagenases were also observed in the soft inflammatory tissues in close association with the cartilage surface, together with variable distributions of mast cells and macrophages. Such observations were generally absent or very much reduced in the control, non-bronchitic specimens. Degenerative changes, atrophy and loss of bronchial cartilage were common features of most chronic bronchitic specimens, this usually being related to intrinsic changes in the chondrocyte phenotype, including proliferative and matrix-degrading properties. Mast cells and macrophages were often observed in close association with the bronchial cartilage, suggesting that inflammatory

cells may also contribute to the mechanisms of bronchial cartilage degradation and loss. These observations of bronchial cartilage degeneration were generally lacking in age-matched non-bronchitic control specimens.

L1 ANSWER 5 OF 33 MEDLINE on STN
ACCESSION NUMBER: 1999267782 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10335301
TITLE: Remobilization does not fully restore immobilization induced articular **cartilage atrophy**.
AUTHOR: Haapala J; Arokoski J P; Hyttinen M M; Lammi M; Tammi M; Kovanen V; Helminen H J; Kiviranta I
CORPORATE SOURCE: Department of Surgery, Kuopio University Hospital, Finland.
SOURCE: Clinical orthopaedics and related research, (1999 May) No. 362, pp. 218-29.
Journal code: 0075674. ISSN: 0009-921X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990618
Last Updated on STN: 19990618
Entered Medline: 19990608
AB The recovery of articular cartilage from immobilization induced atrophy was studied. The right hind limbs of 29-week-old beagle dogs were immobilized for 11 weeks and then remobilized for 50 weeks. Cartilage from the immobilized knee was compared with tissue from age matched control animals. After the immobilization period, uncalcified articular cartilage glycosaminoglycan concentration was reduced by 20% to 23%, the reduction being largest (44%) in the superficial zone. The collagen fibril network showed no significant changes, but the amount of collagen crosslinks was reduced (13.5%) during immobilization. After remobilization, glycosaminoglycan concentration was restored at most sites, except for in the upper parts of uncalcified cartilage in the medial femoral and tibial condyles (9% to 17% less glycosaminoglycans than in controls). The incorporation of 35SO4 was not changed, and remobilization also did not alter the birefringence of collagen fibrils. Remobilization restored the proportion of collagen crosslinks to the control level. The changes induced by joint unloading were reversible at most sites investigated, but full restoration of articular cartilage glycosaminoglycan concentration was not obtained in all sites, even after remobilization for 50 weeks. This suggests that lengthy immobilization of a joint can cause long lasting articular cartilage proteoglycan alterations at the same time as collagen organization remains largely unchanged. Because proteoglycans exert strong influence on the biomechanical properties of cartilage, lengthy immobilization may jeopardize the well being of articular cartilage.

L1 ANSWER 6 OF 33 MEDLINE on STN
ACCESSION NUMBER: 92208668 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1555051
TITLE: A mini review: proteoglycan aggregate profiles in the Pond-Nuki dog model of osteoarthritis and in canine disuse atrophy.
AUTHOR: Howell D S; Muller F; Manicourt D H
CORPORATE SOURCE: Department of Medicine, University of Miami School of Medicine, FL 33101.
SOURCE: British journal of rheumatology, (1992) Vol. 31 Suppl 1, pp. 7-11. Ref: 24
Journal code: 8302415. ISSN: 0263-7103.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)

LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199205
ENTRY DATE: Entered STN: 19920515
Last Updated on STN: 19920515
Entered Medline: 19920507

AB The Pond-Nuki dog model of osteoarthritis has characteristics which seem to mimic the human disease in early stages, particularly with respect to progressive changes in the cartilage matrix. Aggregating proteoglycans were studied using novel extraction and ultracentrifugation methods designed to separate very large macromolecules. With these methods two large peaks of proteoglycan (PG) aggregates (PGA-1 and PGA-2) were separated in preparative amounts and were shown to have unequivocal differences in composition in many respects. The profiles of these peaks have been studied as a function of joint location, topographic site, cartilage layer, presence of **cartilage atrophy** versus osteoarthritis, as well as treatment of the animals with various agents. Both link protein (essential for forming link-protein stabilized aggregates) and hyaluronate are required to regenerate normal aggregate profiles from the deficient aggregate fractions obtained from osteoarthritic cartilage. Canine proteoglycan link-stabilized aggregates (PGA-2) are confined to the middle and deep zone of cartilage. We believe that their reduction or elimination in the Pond-Nuki model results from a disturbance or loss of functional link protein (and hyaluronate), thereby weakening the middle and deep cartilage layers.

L1 ANSWER 7 OF 33 MEDLINE on STN
ACCESSION NUMBER: 90382043 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2205438
TITLE: Pathophysiology of chronic obstructive pulmonary disease.
AUTHOR: Thurlbeck W M
CORPORATE SOURCE: Department of Pathology, University of British Columbia, Vancouver, Canada.
SOURCE: Clinics in chest medicine, (1990 Sep) Vol. 11, No. 3, pp. 389-403. Ref: 78
Journal code: 7907612. ISSN: 0272-5231.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199010
ENTRY DATE: Entered STN: 19901122
Last Updated on STN: 19970203
Entered Medline: 19901026

AB Chronic airflow obstruction (CAO) is a syndrome that is produced by a variety of lesions which may occur in bronchi (large airways), bronchioles (small airways), and lung parenchyma (gas exchanging lung). These lesions frequently occur together in various combinations because of a common etiologic agent, tobacco smoke. Occasionally, one lesion or another may play a dominant role. The major disease of the large airways is chronic bronchitis, or chronic sputum production, and it is defined clinically. Its morphologic counterpart is mucous gland enlargement. Mucous gland enlargement is poorly related to CAO. Other lesions of the large airways--inflammation, smooth muscle hyperplasia, **cartilage atrophy**, and bronchial wall thickening--have also been described, but their functional consequences are uncertain. Bronchiolar lesions are well recognized in CAO, but their relative importance may differ in patients with mild CAO, compared to patients with severe CAO. In mild CAO, inflammation is a very important lesion, and its probable consequences--narrowing, fibrosis, and goblet cell metaplasia--have all been found to be important. In severe CAO, inflammation and fibrosis do not appear to be important, but goblet cell metaplasia, bronchiolar tortuosity, and narrowing do. Emphysema is a subset of airspace enlargement. Emphysema is defined anatomically and is the most important

component of severe CAO. Several forms of emphysema can be recognized morphologically and may have specific clinical associations. However, in the usual patient with severe CAO, it is the severity, rather than the type, of emphysema, that is most significant. The diagnosis of emphysema depends on a combined approach. Significant factors include the clinical history (age, sex, smoking, chronic bronchitis, dyspnea), radiologic evidence of overinflation, and diminished diffusing capacity for carbon monoxide.

L1 ANSWER 8 OF 33 MEDLINE on STN
ACCESSION NUMBER: 90125994 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2404712
TITLE: Pathology of chronic airflow obstruction.
AUTHOR: Thurlbeck W M
CORPORATE SOURCE: University of British Columbia, Faculty of Medicine, Vancouver, Canada.
SOURCE: Chest, (1990 Feb) Vol. 97, No. 2 Suppl, pp. 6S-10S. Ref: 11
Journal code: 0231335. ISSN: 0012-3692.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199003
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19900328
Entered Medline: 19900313

AB Classification of chronic airflow obstruction may be based on the site of the obstructing lesions. It is seldom that only one type of lesion is present, but one may often dominate. In chronic bronchitis, the major disease of large airways, chronic mucus hypersecretion, is reflected by an increase in size of bronchial mucous glands. This may be a factor in airway narrowing, especially with coexisting edema of the airway wall. Excess intralumenal mucus compounds the obstruction. Increased airways reactivity is present in 15 to 70 percent of patients with chronic airflow obstruction. Increased airway muscle and **cartilage atrophy** are features of chronic bronchitis, but the association of increased muscle with increased airway reactivity is poor. Inflammation of the small airways (bronchiolitis) is a significant complication for cigarette smokers and is an important cause of mild chronic airflow obstruction. Goblet cell metaplasia is a reflection of chronic small airways inflammation and, together with intralumenal mucus, is an important feature. Permanent narrowing of the small airways presumably results from inflammation with consequent fibrosis, while functional narrowing results from release of mediators of inflammation. Increased muscle mass is present in some cases. Distortion and irregularity of small airways related to emphysema are major factors in severe obstruction. Lesser degrees of emphysema may be associated with a diminished number of alveolar attachments and mild chronic airflow obstruction. Emphysema, the dominant lesion in patients with severe chronic airflow obstruction, results from parenchymal lesions. Centrilobular emphysema, in which the respiratory bronchioles are selectively or dominantly involved, is the most common form.(ABSTRACT TRUNCATED AT 250 WORDS)

L1 ANSWER 9 OF 33 MEDLINE on STN
ACCESSION NUMBER: 90037565 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2808699
TITLE: Cartilage matrix glycoprotein is present in serum in experimental canine osteoarthritis.
AUTHOR: Fife R S; Brandt K D
CORPORATE SOURCE: Department of Medicine, Indiana University School of Medicine, Indianapolis 46202.
CONTRACT NUMBER: AR-20582 (NIAMS)

AR-34367 (NIAMS)
AR-39250 (NIAMS)
SOURCE: The Journal of clinical investigation, (1989 Nov) Vol. 84, No. 5, pp. 1432-9.
Journal code: 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198912
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19970203
Entered Medline: 19891204

AB We have described previously a disulfide-bonded 550,000-D cartilage matrix glycoprotein (CMGP), which is found in normal hyaline cartilage, fibrocartilage, and the vitreous of the eye, and consists of subunits with apparent molecular weights of 130,000 in 4% gels (116,000 in 9% gels). In osteoarthritic cartilage from dogs subjected to transection of the anterior cruciate ligament (ACL), CMGP is cleaved to major immunoreactive fragments with apparent molecular weights of 65,000 and 75,000 after reduction with 2-mercaptoethanol. In the present study, using immunolocation analysis, a monoclonal antibody to CMGP did not react with serum from 8 of 12 dogs before ACL transection but did react with serum from seven of these animals 4 wk after surgery and with serum from 10 dogs at sacrifice, 8-14 wk after ACL transection. Serum from four dogs reacted with the monoclonal antibody before ACL transection. Serum from two dogs was negative at all time points. Immunolocation studies using a polyclonal antiserum to CMGP were performed in seven of these dogs and produced results identical with the monoclonal antibody in four dogs. In contrast, analysis of serial serum samples from three dogs with **cartilage atrophy** revealed no evidence of CMGP at any time point. These data suggest that CMGP may be a serum marker for osteoarthritis in this canine model.

L1 ANSWER 10 OF 33 MEDLINE on STN
ACCESSION NUMBER: 85045915 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6497604
TITLE: Synovectomy as treatment for purulent joint infection.
AUTHOR: Tscherne H; Giebel G; Muhr G; Howell C
SOURCE: Archives of orthopaedic and traumatic surgery. Archiv fur orthopadische und Unfall-Chirurgie, (1984) Vol. 103, No. 3, pp. 162-4.
Journal code: 7803037. ISSN: 0344-8444.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198412
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19841220

AB Conventional treatment of pyogenic knee joint infections leads to unsatisfactory results. Through early synovectomy, before cartilage damage and osteoarthritis appear, the infected focus can be "excised." Functional after-treatment avoids **cartilage atrophy**, wound adhesions, and muscle weakness. The excellent results after 26 knee joint infections confirm this.

L1 ANSWER 11 OF 33 MEDLINE on STN
ACCESSION NUMBER: 84277727 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6465163
TITLE: Effects of salicylates and other nonsteroidal anti-inflammatory drugs on articular cartilage.
AUTHOR: Brandt K D; Palmoski M J
CONTRACT NUMBER: AM 20582 (NIADDK)

AM 27075 (NIADDK)
SOURCE: The American journal of medicine, (1984 Jul 13) Vol. 77, No. 1A, pp. 65-9.
Journal code: 0267200. ISSN: 0002-9343.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198409
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19840907

AB According to in vivo experimental data, salicylates and several other nonsteroidal anti-inflammatory agents suppress proteoglycan biosynthesis in normal and degenerating articular cartilage. Therapeutic levels of aspirin in vivo had a similar adverse effect on degenerating cartilage, as noted in two canine models of osteoarthritis and **cartilage atrophy**. Because the effective daily antirheumatic dose of nonsteroidal anti-inflammatory drugs is lower than that of salicylates, these drugs may have less negative effects on degenerating articular cartilage. However, clinical significance cannot be extrapolated from these experimental data.

L1 ANSWER 12 OF 33 MEDLINE on STN
ACCESSION NUMBER: 82160255 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7066039
TITLE: Articular **cartilage atrophy** in lower limb amputees.
AUTHOR: Benichou C; Wirotius J M
SOURCE: Arthritis and rheumatism, (1982 Jan) Vol. 25, No. 1, pp. 80-2.
Journal code: 0370605. ISSN: 0004-3591.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198205
ENTRY DATE: Entered STN: 19900317
Last Updated on STN: 19900317
Entered Medline: 19820512

AB A retrospective and radiologic survey of the hips of 53 above-knee amputees showed that none of these hips was normal. Osteoporosis was present in all subjects, and cartilage thickness was reduced in 27 cases. This reduced thickness was inversely correlated with stump length, since it occurred in 11 of 13 upper-third amputees, but in none of 10 lower-third amputees. The mechanisms of **cartilage atrophy** are discussed.

L1 ANSWER 13 OF 33 MEDLINE on STN
ACCESSION NUMBER: 82091306 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7317111
TITLE: Running inhibits the reversal of atrophic changes in canine knee cartilage after removal of a leg cast.
AUTHOR: Palmoski M J; Brandt K D
CONTRACT NUMBER: AM 20582 (NIADDK)
AM 27075 (NIADDK)
SOURCE: Arthritis and rheumatism, (1981 Nov) Vol. 24, No. 11, pp. 1329-37.
Journal code: 0370605. ISSN: 0004-3591.
Report No.: NASA-82091306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Space Life Sciences

ENTRY MONTH: 198202
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19820222

AB The effect of vigorous exercise on the reversibility of canine knee **cartilage atrophy** produced by immobilization of the leg was studied. In comparison to cartilage from the contralateral control knees, cartilage from knees which had been immobilized in a cast for 6 weeks showed an increase in water content and decreases in thickness, Safranin O staining of the matrix, uronic acid content, and net proteoglycan synthesis. In addition, the ability of both newly synthesized (35S) and total tissue proteoglycans to interact with hyaluronic acid to form aggregates was diminished; this was apparently due to an abnormality in the hyaluronate-binding region of the core proteins. If the casts were removed and the animals were then allowed to ambulate ad libitum for 3 weeks, all of these changes were reversed. However, knee cartilage from 3 dogs which had been run daily on a treadmill (6 miles/day) for 3 weeks after removal of the casts exhibited continuing decreases in thickness, Safranin O staining, and uronic acid content (mean 31%), even though net proteoglycan synthesis was increased (mean 16%) in comparison to that in control cartilage from the contralateral (nonimmobilized) knee. Furthermore, the abnormality in both 35S- and total tissue proteoglycans which precluded their interaction with high molecular weight hyaluronic acid persisted. In this respect, the proteoglycans were indistinguishable from those obtained from knee cartilage immediately following 6 weeks in a cast.

L1 ANSWER 14 OF 33 MEDLINE on STN
ACCESSION NUMBER: 81281523 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7271631
TITLE: **Cartilage atrophy** following spinal cord damage.
AUTHOR: Anderson J; Breidahl P
SOURCE: Australasian radiology, (1981 Mar) Vol. 25, No. 1, pp. 98-103.
Journal code: 0047441. ISSN: 0004-8461.
PUB. COUNTRY: Australia
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198110
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19811014

L1 ANSWER 15 OF 33 MEDLINE on STN
ACCESSION NUMBER: 74177067 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4832511
TITLE: **Cartilage atrophy.**
AUTHOR: Pool W H Jr
SOURCE: Radiology, (1974 Jul) Vol. 112, No. 1, pp. 47-50.
Journal code: 0401260. ISSN: 0033-8419.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197407
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19740730

L1 ANSWER 16 OF 33 EMBASE
ACCESSION NUMBER: 2004344653 EMBASE
TITLE: **Cartilage atrophy** in the knees of

patients after seven weeks of partial load bearing.
AUTHOR: Hinterwimmer S.; Krammer M.; Krotz M.; Glaser C.; Baumgart R.; Reiser M.; Eckstein F.
CORPORATE SOURCE: Dr. F. Eckstein, Institute of Anatomy, Paracelsus Priv. Medical University, Strubergasse A2, A5020 Salzburg, Germany. Felix.Eckstein@pmu.ac.at
SOURCE: Arthritis and Rheumatism, (2004) Vol. 50, No. 8, pp. 2516-2520. .
Refs: 16
ISSN: 0004-3591 CODEN: ARHEAW
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 014 Radiology
031 Arthritis and Rheumatism
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 9 Sep 2004
Last Updated on STN: 9 Sep 2004

AB Objective. It is currently unknown whether human cartilage properties change during short periods of partial load bearing. We used a post-ankle fracture model to explore whether changes in cartilage morphology occur in the knee under conditions of partial load bearing. Methods. The knees of 20 patients with Weber type B and type C fractures were examined using magnetic resonance imaging. The first scan was obtained shortly (mean ± SD 3.2 ± 3.0 days) after the injury, and a second scan was obtained 7 weeks later (mean ± SD 50.7 ± 5.5 days). The morphology (mean and maximum thickness, volume, and surface area) of the patellar, tibial, and femoral cartilage was determined from coronal and axial magnetic resonance images (fat-suppressed gradient-echo). Results. Between week 0 and week 7, the cross-sectional area of the quadriceps muscle was reduced by 11% (P<0.001). Changes in the mean (±SD) cartilage thickness ranged from -2.9 ± 3.2% in the patella to -6.6 ± 4.9% in the medial tibia. No significant change in cartilage morphology of the contralateral knee was observed. Conclusion. Results of this study demonstrate that in a post-ankle fracture model of partial load bearing, cartilage morphology in all knee compartments is subject to significant change. Changes in the femorotibial joint exceeded those in the patella, whereas no change was observed in the contralateral knee. These findings raise the question of whether cartilage is mechanically less competent and particularly vulnerable after states of partial or complete immobilization.

L1 ANSWER 17 OF 33 EMBASE
ACCESSION NUMBER: 2003511075 EMBASE
TITLE: Longitudinal Analysis of **Cartilage** **Atrophy** in the Knees of Patients with Spinal Cord Injury.
AUTHOR: Vanwanseele B.; Eckstein F.; Knecht H.; Spaepen A.; Stussis E.
CORPORATE SOURCE: B. Vanwanseele, Laboratory for Biomechanics, ETHZ, Wagistrasse 4, Schlieren CH-8652, Switzerland. vanwanseele@biomech.mat.ethz.ch
SOURCE: Arthritis and Rheumatism, (2003) Vol. 48, No. 12, pp. 3377-3381. .
Refs: 15
ISSN: 0004-3591 CODEN: ARHEAW
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 031 Arthritis and Rheumatism
033 Orthopedic Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jan 2004
Last Updated on STN: 16 Jan 2004

AB Objective. A previous cross-sectional study indicated that the morphology of patellar and tibial cartilage is subject to change after spinal cord injury (SCI). The aim of this study was to perform a longitudinal analysis of **cartilage atrophy** in all knee compartments, including the femoral condyles, in SCI patients over 12 months. Methods. The right knees of 9 patients with complete, traumatic SCI were examined shortly after the injury (mean ± SD 9 ± 4 weeks) and at 6 and 12 months postinjury. Three-dimensional morphology of the patellar, tibial, and femoral cartilage (mean and maximum thickness, volume, and surface area) was determined from coronal and transversal magnetic resonance images (fat-suppressed gradient-echo sequences) using validated postprocessing techniques. Results. The mean thickness of knee joint cartilage decreased significantly during the first 6 months after injury (range 5-7%; $P < 0.05$). The mean change at 12 months was 9% in the patella, 11% in the medial tibia, 11% in the medial femoral condyle, 13% in the lateral tibia, and 10% in the lateral femoral condyle ($P < 0.05$ for all compartments). Conclusion. This is the first report of a longitudinal analysis of **cartilage atrophy** in patients with SCI. These data show that human **cartilage atrophies** in the absence of normal joint loading and movement after SCI, with a rate of change that is higher than that observed in osteoarthritis (OA). A potential clinical implication is that cartilage thinning after SCI may affect the stress distribution in the joint and render it vulnerable to OA. Future studies should focus on whether specific exercise protocols and rehabilitation programs can prevent cartilage thinning.

L1 ANSWER 18 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2003069843 EMBASE
TITLE: Reinforced orbitotemporal lift: Contribution to midface rejuvenation.
AUTHOR: Reno W.T.
CORPORATE SOURCE: Dr. W.T. Reno, Rua Paissandu 368, Guaratingueta, Sao Paulo 12 500 121, Brazil
SOURCE: Plastic and Reconstructive Surgery, (2003) Vol. 111, No. 2, pp. 869-877. .
Refs: 24
ISSN: 0032-1052 CODEN: PRSUAS
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 009 Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20 Feb 2003
Last Updated on STN: 20 Feb 2003

AB The changes in the aging face occur from progressive ptosis of the skin, fat, and muscle, in conjunction with bone absorption and **cartilage atrophy**. In the orbital region, hollowness and compartmentalization occur. Conventional face lift procedures correct only the skin flaccidity, and superficial musculoaponeurotic system techniques reposition the skin and platysma without repositioning the middle third of the face, creating an artificial jawline. Subperiosteal rhytidectomy disrupts the anatomy of the periorbita, which gives the patient a certain scarecrow aspect. Composite rhytidectomy associated with brow lift and blepharoplasty may offer better results, with improvement in the malar and orbital regions. The reinforced orbitotemporal lift (ROTEL) is a new procedure in a face lift that allows the orbicularis oculi muscle and all the structures connected to it to be elevated and stretched and the orbitotemporal skin to be raised, repositioning these structures and ending orbital compartmentalization. The result is an impressive improvement in the malar-orbitotemporal region, resulting in a natural and youthful appearance.

L1 ANSWER 19 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights

reserved on STN
ACCESSION NUMBER: 2000048614 EMBASE
TITLE: Bronchial **cartilage atrophy** in chronic bronchitis: observations on chondrolytic processes.
AUTHOR: Tetlow L.C.; Freemont A.J.; Woolley D.E.
CORPORATE SOURCE: Dr. L.C. Tetlow, University Department of Medicine, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, United Kingdom. lynne.c.tetlow@man.ac.uk
SOURCE: Pathobiology, (1999) Vol. 67, No. 4, pp. 196-201. .
Refs: 22
ISSN: 1015-2008 CODEN: PATHEF
COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
011 Otorhinolaryngology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 17 Feb 2000
Last Updated on STN: 17 Feb 2000

AB The status of bronchial cartilage degeneration in chronic bronchitis is unclear, and little is known about the chondrolytic mechanisms involved. The potential contributions of various inflammatory cells, chondrocytes and cartilage-degrading enzymes to **cartilage atrophy** have been examined. Bronchial cartilage specimens were obtained at autopsy from lobar secondary bronchi from chronic bronchitics and age-matched controls; each was examined by light microscopy and immunohistology for the distributions of mast cells, macrophages, eosinophils, collagenase 1, collagenase 3, and degradation products of cartilage collagen. Most bronchitic specimens showed hypertrophic chondrocytes, some of which were immunostained for collagenase 3, and occasionally for collagenase 1. Evidence for collagen degradation products was demonstrated around the lacunae of a proportion of chondrocytes, and both collagenases were also observed in the soft inflammatory tissues in close association with the cartilage surface, together with variable distributions of mast cells and macrophages. Such observations were generally absent or very much reduced in the control, non-bronchitic specimens. Degenerative changes, atrophy and loss of bronchial cartilage were common features of most chronic bronchitic specimens, this usually being related to intrinsic changes in the chondrocyte phenotype, including proliferative and matrix-degrading properties. Mast cells and macrophages were often observed in close association with the bronchial cartilage, suggesting that inflammatory cells may also contribute to the mechanisms of bronchial cartilage degradation and loss. These observations of bronchial cartilage degeneration were generally lacking in age-matched non-bronchitic control specimens. Copyright (C) 2000 S. Karger AG, Basel.

L1 ANSWER 20 OF 33 EMBASE
ACCESSION NUMBER: 1999173382 EMBASE
TITLE: Remobilization does not fully restore immobilization induced articular **cartilage atrophy**.
AUTHOR: Haapala J.; Arokoski J.P.A.; Hyttinen M.M.; Lammi M.; Markku T.; Kovanen V.; Helminen H.J.; Kiviranta I.
CORPORATE SOURCE: Dr. J. Haapala, Harjukatu 48, FIN-15110 Lahti, Finland
SOURCE: Clinical Orthopaedics and Related Research, (1999) No. 362, pp. 218-229. .
Refs: 39
ISSN: 0009-921X CODEN: CORTBR
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 033 Orthopedic Surgery
LANGUAGE: English

SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 3 Jun 1999
Last Updated on STN: 3 Jun 1999

AB The recovery of articular cartilage from immobilization induced atrophy was studied. The right hind limbs of 29-week-old beagle dogs were immobilized for 11 weeks and then remobilized for 50 weeks. Cartilage from the immobilized knee was compared with tissue from age matched control animals. After the immobilization period, uncalcified articular cartilage glycosaminoglycan concentration was reduced by 20% to 23%, the reduction being largest (44%) in the superficial zone. The collagen fibril network showed no significant changes, but the amount of collagen crosslinks was reduced (13.5%) during immobilization. After rembilization, glycosaminoglycan concentration was restored at most sites, except for in the upper parts of uncalcified cartilage in the medial femoral and tibial condyles (9% to 17% less glycosaminoglycans than in controls). The incorporation of 35SO4 was not changed, and remobilization also did not alter the birefringence of collagen fibrils. Remobilization restored the proportion of collagen crosslinks to the control level. The changes induced by joint unloading were reversible at most sites investigated, but full restoration of articular cartilage glycosaminoglycan concentration was not obtained in all sites, even after remobilization for 50 weeks. This suggests that lengthy immobilization of a joint can cause long lasting articular cartilage proteoglycan alterations at the same time as collagen organization remains largely unchanged. Because proteoglycans exert strong influence on the biomechanical properties of cartilage, lengthy immobilization may jeopardize the well being of articular cartilage.

L1 ANSWER 21 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 95302302 EMBASE
DOCUMENT NUMBER: 1995302302
TITLE: [Pathology of the locomotory system in ankylosing spondylitis].
PATHOMORPHOLOGIE DER BEWEGUNGSORGANE BEI DER SPONDYLITIS ANKYLOSANS.
AUTHOR: Mohr W.
CORPORATE SOURCE: Abteilung Pathologie, Universitat Ulm, Albert-Einstein-Allee 11,D-89081 Ulm, Germany
SOURCE: Aktuelle Rheumatologie, (1995) Vol. 20, No. 5, pp. 162-170.
ISSN: 0341-051X CODEN: AKRHDB
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
031 Arthritis and Rheumatism
LANGUAGE: German
SUMMARY LANGUAGE: English; German
ENTRY DATE: Entered STN: 11 Nov 1995
Last Updated on STN: 11 Nov 1995

AB In the review article pathogenesis and morphology of the different manifestations of ankylosing spondylitis in the locomotory system are described. From the morphological point of view the pathogenesis of the wide spread disease is due to inflammation leading to cartilage and bone destruction. An inflammatory granulation tissue destroys the iliosacral joints, the intervertebral joints and discs and the peripheral joints. The subsequent ossification of the intervertebral discs may lead to the bamboo-spine. The role of a subchondral osteitis in the femoral head and of immobilization on the pathogenesis of **cartilage atrophy** are briefly discussed.

L1 ANSWER 22 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 92124282 EMBASE
DOCUMENT NUMBER: 1992124282

TITLE: A mini review: Proteoglycan aggregate profiles in the Pond-Nuki dog model of osteoarthritis and in canine disuse atrophy.
AUTHOR: Howel D.S.; Muller F.; Manicourt D.H.
CORPORATE SOURCE: Department of Medicine, University of Miami, School of Medicine, PO Box 016960,Miami, FL 33101, United States
SOURCE: British Journal of Rheumatology, (1992) Vol. 31, No. 4 SUPPL., pp. 7-11. .
ISSN: 0263-7103 CODEN: BJRHDF
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 031 Arthritis and Rheumatism
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 15 May 1992
Last Updated on STN: 15 May 1992

AB The Pond-Nuki dog model of osteoarthritis has characteristics which seem to mimic the human disease in early stages, particularly with respect to progressive changes in the cartilage matrix. Aggregating proteoglycans were studied using novel extraction and ultracentrifugation methods designed to separate very large macromolecules. With these methods two large peaks of proteoglycan (PG) aggregates (PGA-1 and PGA-2) were separated in preparative amounts and were shown to have unequivocal differences in composition in many respects. The profiles of these peaks have been studied as a function of joint location, topographic site, cartilage layer, presence of **cartilage atrophy** versus osteoarthritis, as well as treatment of the animals with various agents. Both link protein (essential for forming link-protein stabilized aggregates) and hyaluronate are required to regenerate normal aggregate profiles from the deficient aggregate fractions obtained from osteoarthritic cartilage. Canine proteoglycan link-stabilized aggregates (PGA-2) are confined to the middle and deep zone of cartilage. We believe that their reduction or elimination in the Pond-Nuki model results from a disturbance or loss of function link protein (and hyaluronate), there by weakening the middle and deep cartilage layers.

L1 ANSWER 23 OF 33 EMBASE
ACCESSION NUMBER: 90263666 EMBASE
DOCUMENT NUMBER: 1990263666
TITLE: Pathophysiology of chronic obstructive pulmonary disease.
AUTHOR: Thurlbeck W.M.
CORPORATE SOURCE: Department of Pathology, University of British Columbia, 2211 Westbrook Mall,Vancouver, BC V6T 1W5, Canada
SOURCE: Clinics in Chest Medicine, (1990) Vol. 11, No. 3, pp. 389-403. .
ISSN: 0272-5231 CODEN: CCHMDA
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
006 Internal Medicine
015 Chest Diseases, Thoracic Surgery and Tuberculosis
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991

AB Chronic airflow obstruction (CAO) is a syndrome that is produced by a variety of lesions which may occur in bronchi (large airways), bronchioles (small airways), and lung parenchyma (gas exchanging lung). These lesions frequently occur together in various combinations because of a common etiologic agent, tobacco smoke. Occasionally, one lesion or another may play a dominant role. The major disease of the large airways is chronic bronchitis, or chronic sputum production, and it is defined clinically. Its morphologic counterpart is mucous gland enlargement. Mucous gland enlargement is poorly related to CAO. Other lesions of the large airways

- inflammation, smooth muscle hyperplasia, **cartilage atrophy**, and bronchial wall thickening - have also been described, but their functional consequences are uncertain. Bronchiolar lesions are well recognized in CAO, but their relative importance may differ in patients with mild CAO, compared to patients with severe CAO. In mild CAO, inflammation is a very important lesion, and its probable consequences - narrowing, fibrosis, and goblet cell metaplasia - have all been found to be important. In severe CAO, inflammation and fibrosis do not appear to be important, but goblet cell metaplasia, bronchiolar tortuosity, and narrowing do. Emphysema is a subset of airspace enlargement. Emphysema is defined anatomically and is the most important component of severe CAO. Several forms of emphysema can be recognized morphologically and may have specific clinical associations. However, in the usual patient with severe CAO, it is the severity, rather than the type, of emphysema, that is most significant. The diagnosis of emphysema depends on a combined approach. Significant factors include the clinical history (age, sex, smoking, chronic bronchitis, dyspnea), radiologic evidence of overinflation, and diminished diffusing capacity for carbon monoxide.

L1 ANSWER 24 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 90040671 EMBASE
DOCUMENT NUMBER: 1990040671
TITLE: Pathology of chronic airflow obstruction.
AUTHOR: Thurlbeck W.M.
CORPORATE SOURCE: University of British Columbia, Faculty of Medicine, Vancouver, BC, Canada
SOURCE: Chest, (1990) Vol. 97, No. 2 SUPPL., pp. 6S-10S. .
ISSN: 0012-3692 CODEN: CHETBF
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
015 Chest Diseases, Thoracic Surgery and Tuberculosis
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991

AB Classification of chronic airflow obstruction may be based on the site of the obstructing lesions. It is seldom that only one type of lesion is present, but one may often dominate. In chronic bronchitis, the major disease of large airways, chronic mucus hypersecretion, is reflected by an increase in size of bronchial mucous glands. This may be a factor in airway narrowing, especially with coexisting edema of the airway wall. Excess intralumenal mucus compounds the obstruction. Increased airways reactivity is present in 15 to 70 percent of patients with chronic airflow obstruction. Increased airway muscle and **cartilage atrophy** are features of chronic bronchitis, but the association of increased muscle with increased airway reactivity is poor. Inflammation of the small airways (bronchiolitis) is a significant complication for cigarette smokers and is an important cause of mild chronic airflow obstruction. Goblet cell metaplasia is a reflection of chronic small airways inflammation and, together with intralumenal mucus, is an important feature. Permanent narrowing of the small airways presumably results from inflammation with consequent fibrosis, while functional narrowing results from release of mediators of inflammation. Increased muscle mass is present in some cases. Distortion and irregularity of small airways related to emphysema are major factors in severe obstruction. Lesser degrees of emphysema may be associated with a diminished number of alveolar attachments and mild chronic airflow obstruction. Emphysema, the dominant lesion in patients with severe chronic airflow obstruction, results from parenchymal lesions. Centrilobular emphysema, in which the respiratory bronchioles are selectively or dominantly involved, is the most common form. Familial α1-antiprotease deficiency is the classic example of panacinar

emphysema. When severe, this condition is dominantly a lower zonal disease.

L1 ANSWER 25 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 89272275 EMBASE
DOCUMENT NUMBER: 1989272275
TITLE: Cartilage matrix glycoprotein is present in serum in experimental canine osteoarthritis.
AUTHOR: Fife R.S.; Brandt K.D.
CORPORATE SOURCE: Rheumatology Division, Department of Medicine, Indiana University School of Medicine, Indianapolis, IN 46202, United States
SOURCE: Journal of Clinical Investigation, (1989) Vol. 84, No. 5, pp. 1432-1439. .
ISSN: 0021-9738 CODEN: JCINAO
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 029 Clinical Biochemistry
031 Arthritis and Rheumatism
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 1991
Last Updated on STN: 12 Dec 1991

AB We have described previously a disulfide-bonded 550,000-D cartilage matrix glycoprotein (CMGP), which is found in normal hyaline cartilage, fibrocartilage, and the vitreous of the eye, and consists of subunits with apparent molecular weights of 130,000 in 4% gels (116,000 in 9% gels). In osteoarthritic cartilage from dogs subjected to transection of the anterior cruciate ligament (ACL), CMGP is cleaved to major immunoreactive fragments with apparent molecular weights of 65,000 and 75,000 after reduction with 2-mercaptoethanol. In the present study, using immunolocation analysis, a monoclonal antibody to CMGP did not react with serum from 8 of 12 dogs before ACL transection but did react with serum from seven of these animals 4 wk after surgery and with serum from 10 dogs at sacrifice, 8-14 wk after ACL transection. Serum from four dogs reacted with the monoclonal antibody before ACL transection. Serum from two dogs was negative at all time points. Immunolocation studies using a polyclonal antiserum to CMGP were performed in seven of these dogs and produced results identical with the monoclonal antibody in four dogs. In contrast, analysis of serial serum samples from these three dogs with **cartilage atrophy** revealed no evidence of CMGP at any time point. These data suggest that CMGP may be a serum marker for osteoarthritis in this canine model.

L1 ANSWER 26 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 86101093 EMBASE
DOCUMENT NUMBER: 1986101093
TITLE: Rheumatoid articular pannus. Histogenesis and the mechanism of articular cartilage destruction by pannus.
AUTHOR: Wierzchowska E.; Maldyk E.
CORPORATE SOURCE: Zakladu Anatomii Patologicznej Instytutu Reumatologicznego, Warszawa, Poland
SOURCE: Patologia Polska, (1985) Vol. 36, No. 2, pp. 178-186. .
CODEN: PAPOAC
COUNTRY: Poland
DOCUMENT TYPE: Journal
FILE SEGMENT: 031 Arthritis and Rheumatism
005 General Pathology and Pathological Anatomy
LANGUAGE: Polish
SUMMARY LANGUAGE: English; Russian
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

AB Atricular pannus in 40 women and 10 men with classic or definite

rheumatoid arthritis was examined histopathologically and histochemically. The results of the examinations show that there are two types of pannus: inactive and active one. Inactive pannus is a poorly vascularized coat connective tissue coering the surface of articular cartilage. Active pannus has a connective tissue of loose structure and its cells penetrate deeply into the cartilage in a similar manner as neoplastic cells. In both cases destruction and articular **cartilage atrophy** are the endpoints.

L1 ANSWER 27 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 84207067 EMBASE
DOCUMENT NUMBER: 1984207067
TITLE: Synovectomy as treatment for purulent joint infection.
AUTHOR: Tscherne H.; Giebel G.; Muhr G.; Howell Ch.
CORPORATE SOURCE: Unfallchirurgische Klinik der Medizinischen Hochschule, D-3000 Hannover 61, Germany
SOURCE: Archives of Orthopaedic and Traumatic Surgery, (1984) Vol. 103, No. 3, pp. 162-164. .
CODEN: AOUNAZ
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 033 Orthopedic Surgery
031 Arthritis and Rheumatism
004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: German
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

AB Conventional treatment of pyogenic knee joint infections leads to unsatisfactory results. Through early synovectomy, before cartilage damage and osteoarthritis appear, the infected focus can be 'excised'. Functional after-treatment avoids **cartilage atrophy**, wound adhesions, and muscle weakness. The excellent results after 26 knee joint infections confirm this.

L1 ANSWER 28 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 84175147 EMBASE
DOCUMENT NUMBER: 1984175147
TITLE: Effects of salicylates and other nonsteroidal anti-inflammatory drugs on articular cartilage.
AUTHOR: Brandt K.D.; Palmoski M.J.
CORPORATE SOURCE: Rheumatology Division, Indiana University Medical Center, Indianapolis, IN 46223, United States
SOURCE: American Journal of Medicine, (1984) Vol. 77, No. 1 A, pp. 65-69. .
CODEN: AJMEAZ
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
031 Arthritis and Rheumatism
033 Orthopedic Surgery
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

AB According to in vivo experimental data, salicylates and several other nonsteroidal anti-inflammatory agents suppress proteoglycan biosynthesis in normal and degenerating articular cartilage. Therapeutic levels of aspirin in vivo had a similar adverse effect on degenerating cartilage, as noted in two canine models of osteoarthritis and **cartilage atrophy**. Because the effective daily antirheumatic dose of nonsteroidal anti-inflammatory drugs is lower than that of salicylates, these drugs may have less negative effects on degenerating articular

cartilage. However, clinical significance cannot be extrapolated from these experimental data.

L1 ANSWER 29 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 82053924 EMBASE
DOCUMENT NUMBER: 1982053924
TITLE: Articular **cartilage atrophy** in lower limb amputees.
AUTHOR: Benichou C.; Wirotius J.M.
CORPORATE SOURCE: Cent. Hosp. St Cloud, 92210 St Cloud, France
SOURCE: Arthritis and Rheumatism, (1982) Vol. 25, No. 1, pp. 80-82. .
CODEN: ARHEAW
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 031 Arthritis and Rheumatism
033 Orthopedic Surgery
005 General Pathology and Pathological Anatomy
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB A retrospective and radiologic surveys of the hips of 53 above knee amputees showed that none of these hips was normal. Osteoporosis was present in all subjects, and cartilage thickness was reduced in 27 cases. This reduced thickness was inversely correlated with stump length, since it occurred in 11 of 13 upper third amputees, but in none of 10 lower third amputees. The mechanisms of **cartilage atrophy** are discussed.

L1 ANSWER 30 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 82005334 EMBASE
DOCUMENT NUMBER: 1982005334
TITLE: Running inhibits the reversal of atrophic changes in canine knee cartilage after removal of a leg cast.
AUTHOR: Palmoski M.J.; Brandt K.D.
CORPORATE SOURCE: Rheumatol. Div., Indiana Univ. Sch. Med., Indianapolis, IN 46223, United States
SOURCE: Arthritis and Rheumatism, (1981) Vol. 24, No. 11, pp. 1329-1337. .
CODEN: ARHEAW
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 031 Arthritis and Rheumatism
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB The effect of vigorous exercise on the reversibility of canine knee **cartilage atrophy** produced by immobilization of the leg was studied. In comparison to cartilage from the contralateral control knees, cartilage from knees which had been immobilized in a cast for 6 weeks showed an increase in water content and decreases in thickness, Safranin O staining of the matrix, uronic acid content, and net proteoglycan synthesis. In addition, the ability of both newly synthesized (35S) and total tissue proteoglycans to interact with hyaluronic acid to form aggregates was diminished; this was apparently due to an abnormality in the hyaluronate-binding region of the core proteins. If the casts were removed and the animals were then allowed to ambulate ad libitum for 3 weeks, all of these changes were reversed. However, knee cartilage from 3 dogs which had been run daily on a treadmill (6 miles/day) for 3 weeks after removal of the casts exhibited continuing decreases in thickness, Safranin O staining, and uronic acid content (mean 31%), even though net proteoglycan synthesis was increased (mean 16%) in comparison to that in control cartilage from the contralateral

(nonimmobilized) knee. Furthermore, the abnormality in both 35S- and total tissue proteoglycans which precluded their interaction with high molecular weight hyaluronic acid persisted. In this respect, the proteoglycans were indistinguishable from those obtained from knee cartilage immediately following 6 weeks in a cast.

L1 ANSWER 31 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 81232043 EMBASE
DOCUMENT NUMBER: 1981232043
TITLE: Effect of joint disuse and subsequent exercise on proteoglycan mtabolism and aggregation in articular cartilage.
AUTHOR: Palmoski M.; Brandt K.
CORPORATE SOURCE: Indiana Univ. Sch. Med., Indianapolis, IN, United States
SOURCE: Seminars in Arthritis and Rheumatism, (1981) Vol. 11, No. 1 Suppl. 1, pp. 30-31. .
CODEN: SAHRBF
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 031 Arthritis and Rheumatism
019 Rehabilitation and Physical Medicine
029 Clinical Biochemistry
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB We have described changes in knee cartilage after amputation of the ipsilateral paw identical to changes produced by immobilization. Notably, the cartilage degeneration in the paw transection model developed in the presence of a normal arc of knee movement, strongly suggesting that the changes arising with immobilizaton were not due simply to a lack of joint motion but to reduction in the loading of the cartilage, which results from contraction of the muscles that span the joint and stabilize the limb instance. The objective of the present study was to determine whether the reversibility of the **cartilage atrophy** induced by immobilization might be affected by vigorous but physiologic usage of the recently constrained joint.

L1 ANSWER 32 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 81190509 EMBASE
DOCUMENT NUMBER: 1981190509
TITLE: **Cartilage atrophy** following spinal cord damage.
AUTHOR: Anderson J.; Breidahl P.
CORPORATE SOURCE: Dept. Diagn. Radiol., Roy. Perth Hosp., Perth, WA, Australia
SOURCE: Australasian Radiology, (1981) Vol. 25, No. 1, pp. 98-103. .
CODEN: AURDAW
COUNTRY: Australia
DOCUMENT TYPE: Journal
FILE SEGMENT: 014 Radiology
033 Orthopedic Surgery
008 Neurology and Neurosurgery
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB A retrospective analysis of hip joint space measurement of patients who had previously sustained spinal cord damage was undertaken. This paper confirms previous findings that patients with flaccid paralysis in lower limbs develop hip joint space narrowing. It has been found that this phenomenon is far more frequent than previously reported and can be seen in a significant proportion of patients with lower limb spasticity.

L1 ANSWER 33 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 75065794 EMBASE
DOCUMENT NUMBER: 1975065794
TITLE: **Cartilage atrophy.**
AUTHOR: Pool Jr W.H.
CORPORATE SOURCE: Dept. Radiol., Med. Coll. Georgia, Augusta, Ga., United States
SOURCE: Radiology, (1974) Vol. 112, No. 1, pp. 47-50. .
CODEN: RADLAX
DOCUMENT TYPE: Journal
FILE SEGMENT: 014 Radiology
031 Arthritis and Rheumatism
033 Orthopedic Surgery
LANGUAGE: English

AB Two hundred cases of flaccid paralysis of the lower extremities were reviewed, and in 25 the cartilaginous space of the hip joint was found to be narrowed by at least 50%. It is postulated that the cartilage reduction is the result of atrophy following an altered nutrition associated with lack of stress and a decrease in the production of synovial fluid.

=>
=> logoff h

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	106.77	106.98

SESSION WILL BE HELD FOR 60 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 11:44:53 ON 17 APR 2006